

REVIEW ARTICLE

Exercise and sleep

Helen S. Driver¹ and Sheila R. Taylor²

¹*Department of Psychiatry, University of Toronto, University Health Network,
Toronto Western Hospital Applied and Interventional Research Division, Canada and*

²*Department of Physiology, University of the Witwatersrand, Johannesburg, South Africa*

This paper reviews the literature on the association between exercise and sleep. The epidemiological and experimental evidence for whether or not acute and chronic exercise promote sleep is discussed, as well as moderating factors and agendas for future directions of study. The expectation that exercise will benefit sleep can partly be attributed to traditional hypotheses that sleep serves energy conservation, body restoration or thermoregulatory functions, all of which have guided much of the research in this field. Exercise is a complex activity that can be beneficial to general well-being but may also stress the body. Differences in the exercise protocols studied (e.g. aerobic or anaerobic, intensity, duration) and interactions between individual characteristics (e.g. fitness, age and gender) cloud the current experimental evidence supporting a sleep-enhancing effect of exercise. In addition, the tendency to study changes in small groups of good sleepers may also underestimate the efficacy of exercise for promoting sleep. Although only moderate effect sizes have been noted, meta-analytical techniques have shown that exercise increased total sleep time and delayed REM sleep onset (10 min), increased slow-wave sleep (SWS) and reduced REM sleep (2–5 min). The sleep-promoting efficacy of exercise in normal and clinical populations has yet to be established empirically.

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Introduction

Exercise is endorsed by the American Sleep Disorders Association and, in most discussions regarding sleep hygiene, is considered a non-pharmacological intervention to improve sleep [1,2]. Large epidemiological surveys show that the therapeutic and sleep-promoting benefits of moderate, regular physical activity are accepted by the general public [3,4], but empirical evidence is not so compelling [5–8]. This dichotomy between general beliefs and empirical evidence is not unique to the sleep literature [9]. The paradox between expectations and experimental evidence from sleep studies may be attributed to a number of factors, including differing methodology, zero or inadequate control groups, small sample sizes, a predominant focus on good sleepers as study participants, and an impetus for the research to test various theories of sleep function [6,7].

Correspondence to be addressed to: Helen Driver, Department of Psychiatry, Toronto Western Hospital (ECW3D-032), 399 Bathurst Street, Toronto, Ontario M5T 2S8, Canada. Tel: +1 416 603 5765; Fax: +1 416 603 5292; E-mail: hdriver@uhnres.utoronto.ca

The question of the sleep-promoting efficacy of exercise is quite recent and is difficult to answer from the current literature. To expect a straightforward answer is an oversimplification of a complex set of activities that may be physiologically and psychologically beneficial but may also stress the body. In order to show that an intervention improves sleep, waking feeling refreshed should accompany changes such as a shorter time taken to fall asleep (sleep onset), fewer awakenings and less time spent awake. The changes typify the therapeutic responses that are sought for effective treatment of insomnia [10]. Unfortunately, to date, the exercise studies have largely focused on good sleepers, leaving little room for sleep improvement (ceiling effect), whether this is assessed subjectively or according to polysomnographic observations. Furthermore, when evaluating the effect of either acute or chronic exercise on sleep, issues of temperature regulation, possible interactions with other systems (cardiorespiratory, immune and endocrine) as well as mood need to be considered.

This review will begin with an assessment of theoretical models that have driven much of the early research regarding the effects of exercise on sleep. Thereafter, the epidemiological and experimental evidence that acute and chronic exercise promote sleep will be discussed, as well as moderating factors and agendas for future directions in this field of study.

Theoretical models that have predicted positive effects of exercise

Interpretations of some traditional theories of sleep function, which include the thermoregulatory [11], body restoration [12] and energy conservation hypotheses [13], have led to predictions that exercise will have uniquely potent sleep-promoting effects.

Thermogenic hypothesis

Some evidence suggests that a “trigger” for sleep onset is the evening decline in body temperature, which is primarily mediated by increased peripheral skin blood flow [14]. Sleep onset is associated with peripheral heat dissipation through vasodilation [15] and increased sweating, together with a reduction in metabolic rate and core body temperature during sleep [16]. In addition to the nocturnal circadian decline, body temperature is regulated at a lower level during non-rapid eye movement (nonREM) sleep than during wakefulness while thermoregulation is inhibited during rapid eye movement (REM) sleep [16]. The anterior hypothalamus plays a crucial role in sleep and temperature regulation [11], so it has been postulated that body temperature elevation before bedtime can activate both heat-loss and the associated sleep mechanisms [11,17,18]. Changes in body or brain temperature have also been found to influence the sleep EEG power spectrum [19]. A thermoregulatory control hypothesis has been proposed based on observations of increased sleep after waking heat loads, waking hypothermia after sleep deprivation and the close coupling between circadian rhythms in sleep and body temperature [11]. McGinty and Szymusiak [11] suggest that brain and body cooling, induced during sleep, and slow wave sleep (SWS, stages 3 and 4 of nonREM sleep) in particular, provide several adaptations, including lower energy utilization, reduced cerebral metabolism, protection of the brain against sustained high temperatures of wakefulness, and facilitation of immune defence processes.

The thermogenic hypothesis predicts a unique role for exercise, which raises body

temperature more readily than any other stimulus. Body temperature increases are proportional to the workload, such that exercising at 70% of maximal oxygen utilization (VO_2max) would raise the temperature by approximately 2°C after 15–20 min of continuous work at a constant workload during a 60 min work period [20]. Evidence for a thermogenic effect of exercise on sleep was provided by Horne and Moore [17]. They compared the effects of exercise, of similar intensity and duration, in which body temperature elevation was potentiated by wearing extra clothes (hot) or blunted via body cooling; only exercise in the hot condition elicited increases in SWS. However, other studies where body temperature was elevated by exercise of moderate-intensity (60% VO_2max) for 1 h [21] or 3 h at 70% VO_2max [22] reported no effect on sleep architecture. Neither was sleep altered when body temperature was raised in response to the thermic effect of food [23]. It is unlikely that alterations in sleep following exercise are largely in response to the thermal challenge particularly in good sleepers, but, as discussed below, temperature effects could play a greater role in poor sleepers.

Clinically, an association between temperature downregulation and sleep is suggested by findings that several groups with impaired sleep—including insomniacs, depressed patients and older individuals—have a concomitant impairment in nocturnal temperature downregulation [16]. Some evidence suggests that daytime responsiveness to a thermal challenge is associated with the ability to thermoregulate during sleep [24]. The putative soporific effects of melatonin have also been ascribed to its hypothermic effect, which is correlated with heat-loss responses (e.g. skin blood flow) to a standard heat stimulus [25]. If sleep problems indeed result from inadequate temperature downregulation, they might be amenable to rapid improvement via heat acclimation following repeated exercise or passive heating. Thermoregulatory mechanisms and homeothermy, or the maintenance of body temperature within certain limits, also features in the two theories of sleep function discussed below.

Energy conservation and body restoration

Traditional hypotheses suggest that sleep, by reducing metabolic requirements, may have an energy conservation and/or tissue restoration function. Both the energy conservation [13] and restorative theories [12] posit that sleep duration and the amount of SWS will increase as a function of increased energy expenditure, thus raising the concept that exercise would have a major impact on sleep, since no other stimulus can so readily deplete energy stores.

There are two versions of the energy conservation theory. One is that sleep is for the reduction of energy expenditure below the level attainable by rest alone [13], the second suggests that sleep sets a limit on metabolic expenditure to the extent necessary to balance a species' energy budget [26]. Evidence contradicting a primary energy conservation function for sleep is that energy expenditure is only 10–15% less than that associated with quiet wakefulness [27], SWS increases during prolonged bed-rest [28] but decreases with restriction of energy intake and weight loss [29]. The alternative view considers sleep to enforce rest and keep energy expenditure at an affordable level [26]. Since small endothermic animals expend much more energy during wakefulness compared with larger endotherms, such as humans, they might require more sleep to maintain homeothermy. The findings of a negative correlation (-0.53) of total daily sleep time and body weight, and a positive correlation (0.37)

with weight-specific metabolic rate, across 84 animal species, support a greater requirement for sleep in species with low energy reserves [26]. However, when statistically controlling for body weight, sleep time was negatively correlated (-0.28) with weight-specific metabolic rate [26] which suggests that the differences in sleep between small and large mammals might be explained by factors other than, or additional to, size. Thus correlational studies of total daily sleep quotas with body weight and metabolic rate fall short of explaining much of the variance in sleep quotas but indicate that sleep has some role in energy regulation [26]. The interspecies variation in daily mammalian sleep quotas is also not explained by the restorative theory.

The body restoration or compensatory theory predicts that conditions for anabolic activity during sleep will be favoured following high catabolic activity during the waking period [12]. According to this hypothesis, sleep should allow for recuperation from body "wear and tear" during wakefulness. Given the metabolism of fuel and use of the body that is required for physical work, exercise has been studied as a stress that should facilitate sleep. A subjective description of sleep as being non-restorative with low energy during the daytime is sometimes used in complaints of insomnia [10]. The subjective complaint of reduced sleep quality, duration, or efficiency in insomniacs may, or may not, be corroborated by objective evidence. An aspect of the restorative theory of sleep relates to the depth of sleep. For more restful sleep, there is some debate as to whether the amount of deep sleep (SWS) and increased electroencephalogram (EEG) slow-wave activity (SWA, in the frequency range from 0.25 to 4.5 Hz), is indicative of good sleep quality and could be another indication of improved sleep [30]. There may also be a delay in the onset of REM sleep (REM latency), possibly decreased REM sleep time, and longer sleep duration. As reviewed below, exercise studies to date have generally shown that the effects of exercise on sleep are quite modest, and can even be disruptive with very long (over 2 h) high-intensity exercise.

Experimental studies showing associations between exercise and sleep

Both the energy conservation and body restoration hypotheses for the function of sleep propose an increased need for sleep following exercise. Small effects of acute exercise on sleep have been reported in recent meta-analytical reviews [7,31]. An advantage of meta-analysis over narrative reviews is that it provides an objective method for combining results from independent studies in a systematic, statistical procedure [7,31]. In the two recent reviews, effect sizes (ES) were calculated by subtracting the mean for the control condition (μ_{controls}) from the mean following exercise (μ_{exercise}) and dividing this quantity by the standard deviation (SD) for the control condition [7] or the pooled SD [31] $(\mu_{\text{exercise}} - \mu_{\text{control}}) / \text{SD}_{\text{control or pooled}}$. Statistical guideposts for judging effects as small, moderate and large were 0.2, 0.5 and 0.8, respectively [7].

In the studies included in the meta-analyses, exercise type was generally aerobic, but the duration, intensity and time of day when performed was highly variable. Exercise consistently increased total sleep time (TST) with ES between 0.31 [31] and 0.41 [7] and delayed REM latency (ES of 0.29 [31] and 0.52 [7] respectively) compared to control conditions; the mean magnitude of the response for both sleep measures was about 10 min [7,31]. Small positive effects (ES about 0.22) in response to acute exercise were noted for SWS [7,31] giving a mean increase of about 4 min [7], with a larger effect size (0.75) on stage 4 sleep alone [31]. For REM sleep, negative ES following exercise ranged from -0.14 [31] to -0.49 [7], with a median decrease of 6 min from control conditions [7].

Overall these effects were modest and were probably influenced by small sample sizes (usually nine participants) consisting of “good sleepers”, and different research protocols. Other potential factors to consider include fitness levels, the proximity of exercise to sleep, age, gender, body mass, and the intensity, duration, timing and type of exercise. Factors known to influence sleep—including circadian rhythms, general health status and exposure to bright light—may also modulate the association between exercise and sleep. Perhaps, given the focus on good sleepers and fit athletes—about half the studies have been conducted with fit athletes [7]—in studies on the effects of exercise on objective measures of sleep [6,7,31], good sleepers may already have optimal sleep, leaving little room for improvement (ceiling effect). Indeed, physical exercise has been found to be beneficial in clinical conditions where poor sleep is a complaint, such as depression [32,33], and decreased the respiratory disturbance index (RDI) in sleep apneics [34]. However, in this group of 11 patients with mild to severe sleep apnea, a 6-month physical training period did not result in any significant changes in their sleep [34]. Where improvements in sleep occur in these patient populations, they may be secondary to decreases in the RDI or depression rather than exercise *per se*.

Assessing the effect of exercise on sleep in healthy individuals may minimize confounding factors, but also limit the potential for improving sleep. Although the effects may be small to moderate, in physically fit individuals exercise seems to influence sleep in the predicted manner increasing TST and prolonging the REM latency (mean difference from control conditions of 10 min), decreasing REM sleep and increasing SWS (by between 2 to 5 min from control conditions) [6,7,30,31,35,36]. Finally, as noted by Kubitz *et al.* [31], the effects of exercise on sleep are comparable with other health-related interventions that have been considered to be effective (ES range 0.24 to 0.8), including the anxiety-reducing effect of exercise (ESs = 0.24–0.56) [For references see 31].

Most studies on the effect of exercise on objective sleep measures have:

1. Tested traditional hypotheses of sleep function such as the thermoregulatory control, energy conservation and body restitution hypotheses.
2. Generally included only “good sleepers” or fit athletes, hence a drawback to extrapolating findings to clinical populations is that these “good sleepers” may already have optimal sleep, allowing little room for improvement.

Epidemiological studies

Objective laboratory studies have been criticized for the small sample sizes and differing protocols. Epidemiological data, while based on self-reports, have the advantage of assessing larger samples. These studies have consistently supported the view that acute and chronic exercise promotes sleep. For example, men and women (36–50 years) in Finland ($n=1190$) listed exercise as the most important sleep-promoting factor when asked open-ended questions about perceived effects of exercise on sleep [4]. Respondents who reported exercising regularly had less daytime tiredness compared with those who were more sedentary. Moreover, 43% of those who reported increased amounts of exercise during the previous 3 months ($n=81$) reported improved subjective sleep, compared with only 1% whose sleep deteriorated. Conversely, 30% of those subjects who reported decreased exercise during the previous 3 months ($n=73$) had

a deterioration in their sleep, whereas only 4% reported improved sleep. More recently, a study of randomly selected women ($n=403$) and men ($n=319$) living in Arizona found a significantly lower prevalence of self-reported sleep problems and daytime sleepiness in physically-active compared to sedentary-respondents [3].

Considering the available epidemiological evidence, there are other factors that should be considered that might influence both exercise and sleep, for example motivation, mood, lifestyle, general well-being, etc. It is plausible that those who sleep better are less tired and fatigued during the day and, therefore, more willing to engage in regular exercise [21]. For example, a study by Suskin *et al.* [37] showed that decreased sleep associated with shift-work rotation resulted in reductions in physical activity and aerobic fitness. Furthermore, physically active individuals may be more likely to engage in other healthy habits conducive to good sleep such as limiting intake of alcohol and caffeine [8]. In the clinical arena, there is a strong emphasis on self-reports for diagnosing and treating insomnia and subjective sleep reports might provide sufficient evidence of improvements to warrant advising a trial of physical exercise to improve not only sleep, but also health and well-being.

Effects of acute exercise on sleep

After exercising, feelings of fatigue may be perceived as sleepiness and thus may be interpreted as facilitating sleep. Fatigue is a common complaint in athletes in training and many patients with sleep disorders. The true genesis of fatigue is not understood, but although fatigue is often accompanied by daytime sleepiness, these two states are not synonymous [38]. Studies that have controlled for napping have generally failed to find a subsequent increase in sleepiness following exercise compared with control conditions [39]. Indeed, exercise results in a transient decrease in sleepiness that is intensity-dependent [39,40] and most pronounced when exercise is performed in the middle of the night [8].

Exercise intensity, type of exercise and the timing in relation to sleep are important contributing factors to the sleep response. It has been suggested that a beneficial effect of exercise may only occur when exercise is conducted close enough to sleep to stimulate the thermoregulatory response [5,17,18,42] but not too close to bedtime to have an alerting or disruptive effect [6]. General sleep hygiene recommendations have been to exercise 5–6 h before bedtime but not closer than 3 h [10]. The exercise should also be of relatively long duration at high rates of energy expenditure [5] and, in the case of fit individuals, lasts for about half an hour [41]. Where significant moderating effects were noted, they were small to moderate—between 6 to 37% of the variance were accounted for by moderating effects [31]. For example, the individual's age and fitness, and the type and duration of exercise accounted for at least 25% of the variance in ESs [31]. The influence of moderating variables on sleep are reviewed below.

Intensity and duration of acute exercise

A common limitation of the meta-analysis procedure is that the data are often inadequate for accurately assessing interactions between moderator variables [7]. However, Youngstedt *et al.* [7] found that the duration of exercise was a more consistent moderator variable than other factors considered, such as fitness and time of day. The most reliable effects,

specifically increased TST and REM latency, and decreased REM sleep, were only observed for exercise lasting more than 1 h—possibly because these were more clearly described in experimental protocols than other conditions such as fitness and time of day. Also, because comparatively fit populations were studied, it is possible that longer exercise durations were needed in order to stimulate a sleep response.

High intensity (75–80% VO_2max for 1.2 h) [17,42] and moderate exercise (50–70% VO_2max for an average of 2.5 h) to exhaustion [43] have both resulted in increased SWS compared to a rest day. In contrast, 1 h of exercise at various exercise intensities up to a workload close to maximum [44] or exercise at a normal training intensity [45] did not effect SWS.

Long distance athletic or endurance events have been used to examine the effects of high intensity and prolonged exercise on recovery sleep [46–49]. Some of these events, however, took place outdoors, in uncontrolled environmental conditions and were competitive events. Realistic competitive situations rather than controlled or counterbalanced laboratory studies introduce confounding factors such as nervous anticipation, increased sympathetic activation and waking early before the race, that may impact on sleep and therefore make it difficult to draw conclusions regarding the effects of exercise alone. On the other hand, a controlled laboratory situation and long distance running is not necessarily the exercise mode of choice for a population seeking a regular exercise routine. While studies after exercising outdoors or following participation in competitive activities may not provide clean laboratory-driven data, they provide valuable insight into recovery from these field-situations and point to the robustness of sleep. However, it should be borne in mind that many other factors, some of which are highlighted at the end of this review, come into play, and the usefulness of extrapolating from these very unusual exercise regimens is limited [5].

Long distance events with the longest post-competition TST were marathon and ultra-marathon competitions. Recordings were not made on the night before the event so that the influence of sleep restriction was not controlled for, but *ad libitum* sleep was permitted after the race [7]. Notwithstanding the lack of laboratory-controlled studies, endurance events elicit SWS enhancement, longer REM latency, decreased REM sleep and increased wakefulness after sleep onset (WASO) [6]. Long distance outdoor events that resulted in increased SWS included a 92 km [46] and a 30–42 km marathon resulting in significantly increased EEG power density in the slow-wave frequency but no change in SWS duration [47]. Whereas these effects were reported for young athletes (22 years and 30–35 years, respectively), older subjects (41 years) who participated in a 42.2 km marathon had a significant post-competition decrease in SWS [48]. The different effects on SWS may be ascribed to different exercise intensities, exercise duration, the time before sleep when exercise was completed and the age difference between the groups. More generally, following a marathon there is evidence of sleep disruption with increased wakefulness [46,48] and possibly a prolonged REM latency [47,49] and decreased REM sleep [47].

The relevance of the effects of moderate and endurance exercise on REM sleep are not clear. The decreased REM sleep propensity may be due to an increased need for nonREM sleep or alternatively an indicator of sleep fragmentation [49]. In a clinical application, it has been proposed that antidepressant effects may be related to the ability of medications to suppress REM sleep [50]. It is tempting therefore to extrapolate that, given the longer REM latency and reduced REM sleep time following participation in outdoor long-distance exercise, physical activity may have antidepressant effects with concomitant improvements in sleep [7,18,21].

Finally, sleep disruptions have been reported in more extreme situations such as following high-intensity exercise exceeding 2 h in duration [49]. Anecdotal accounts and some experimental evidence also indicate that chronically intense levels of exercise or “overtraining” can disturb sleep [51]. Although the focus here has largely been on the beneficial effects of exercise on sleep, there seems to be a threshold level of exercise beyond which sleep is disrupted. Given the effects of prolonged and/or high intensity exercise on REM sleep, this may be a more sensitive index of exercise-induced stress than SWS. Preferably, more sensitive EEG markers such as those obtained using computerized quantitative EEG analyses (e.g. SWA and sleep spindles) may be better indices of sleep quality.

Practice Points

1. Meta-analytical techniques have been employed to resolve issues of low statistical power, differences in protocols and the lack of consensus as to the acute effects of exercise on sleep [7,31]. Although only moderate effect sizes were noted, exercise increased SWS, reduced REM sleep and delayed REM latency in already fit subjects.
2. The duration of exercise was a more consistent moderator variable on the acute effects of exercise on sleep than other factors considered including fitness and time of day, with the most reliable effects only observed following exercise lasting more than 1 h in physically fit individuals.

Research Agenda

Given the longer REM latency and reduced REM-sleep time following participation in outdoor long-distance exercise, physical activity may have antidepressant effects. The effects of acute and chronic exercise on mood in depressed patients and poor sleepers need to be examined.

Type of exercise

The above studies have examined the effect of endurance and aerobic exercise on sleep patterns, but what of the effects of non-aerobic exercise? The type of training reportedly has significant effects on sleep patterns [5,52] and the type of exercise accounts for a small to moderate effect for acute exercise [31]. Non-aerobic exercise such as power training develops different physiological attributes to training programmes that are primarily aerobic, but the effect of this type of exercise on sleep has not been adequately considered. In their study on fit power lifters Montgomery *et al.* [53] found that afternoon exercise did not affect sleep. However, compared to power-trained athletes, endurance athletes had higher levels of SWS, increased sleep duration and shorter sleep onset [5,52]. More recently, 10-weeks of weight-lifting exercise for approximately 1 h 3 days a week, in a group of depressed elderly (>60 years) men and women was found to improve self-reported sleep quality compared to a control group engaged in a health-education program [32]. This benefit to sleep was in addition to improvements in depression measures and quality of life, which may have influenced sleep rather than just the exercise. Although it is not clear whether the exercise *per se*, or the additional factors such as mood

and quality of life improved sleep, the clinical benefit of the high-intensity progressive resistance training exercise to these elders was significant.

Time of day

The time of day when exercise is performed is one of the most consistent moderator variables of the acute effects of exercise on sleep [7,31]. These effects are consistent with common sleep–hygiene recommendations that exercise in the late afternoon—as opposed to exercising late in the evening—will enhance sleep while the latter will impair sleep. However, a recent study by Youngstedt *et al.* [22] did not find that vigorous exercise for 3 h performed late at night, 30 min before bedtime, had any impact on sleep in highly fit male cyclists. There is emerging evidence that exercise has the ability to induce circadian phase-shifting that might be as potent as bright light effects [54].

Gender

Very few studies on exercise and sleep have included women as study participants. A meta-analysis of acute and chronic exercise demonstrated a larger impact on sleep in women [31] than in men. Although this is an encouraging finding given that women appear to suffer from insomnia more than men [55], only four studies included women compared to 26 studies with men. In studies that included men and women as participants, gender differences in the response of sleep to exercise however are not consistent. Following maximal capacity exercise, Bunnell *et al.* [43] found that there was a tendency for a greater increase in SWS in women than in men, but Montgomery *et al.* [53] that SWS decreased in women but not in men after exercise in the afternoon. Furthermore, studies that have included women have not controlled for menstrual cycle phase, oral contraceptive use or menopausal status.

Research Agenda

Considering the variation in body temperature and sleeping metabolic rate across the menstrual cycle [55], controlled studies on the effects of exercise in the follicular and luteal phases are required.

Age

With increasing age, dissatisfaction with sleep quality becomes more common [56], and both SWS and REM sleep decrease regardless of fitness status [57]. Moderate-intensity endurance training in older sedentary men and women with moderate sleep complaints [58] was found to subjectively improve sleep quality. There may be more “room for improvement” in sleep of older individuals. Age has been found to account for as much of the variance in meta-analytical studies as did fitness, and the type and duration of exercise [31]; effect sizes were larger for older than for younger individuals.

Effects of chronic exercise on sleep

Habits that are detrimental to physical health and well-being such as smoking, caffeine, alcohol intake and overeating often complicate an existing sleep problem or hinder treatment [10]. By engaging in regular aerobic exercise these individuals may be more likely to abandon these detrimental habits. Fitness as a moderating factor on sleep has been investigated by examining the effect of improved cardiorespiratory fitness with participation in physical training programmes, enforced non-training and overtraining in athletes and by comparing physically fit athletes to unfit individuals.

Improved fitness for optimal sleep?

Improvements in self-rated sleep quality have been found following 16-weeks of moderate-intensity endurance training in older (50–76 years) sedentary men and women who reported moderate sleep complaints [58]. These effects were not evident after only 8 weeks, suggesting that the training must be of sufficient duration. In a large survey of middle-aged to elderly subjects, a reduced likelihood of having difficulty maintaining sleep and of having a sleep complaint was associated with regular activity at least once a week [3]. In the study by Singh *et al.* [32] of older, depressed patients mentioned earlier, self-reported sleep quality was significantly improved following 10-weeks of weight training when compared with health education training. Again, the possible role of improved quality of life as a contributing factor should be borne in mind.

Exercise has been considered as a possible treatment for psychophysiological insomnia; sleep hygiene education and a 4-week exercise program resulted in small improvements in sleep, with longer TST, decreased sleep latency and WASO, as measured using actigraphy [59]. Since the exercise intervention was not administered alone, the sleep effects may be partially attributed to the accompanying improved sleep hygiene. However, with sleep hygiene alone, there was no improvement in sleep, which suggests a place for the role of exercise to improve sleep in this patient population. Since it is the perception of poor sleep quality that motivates people to seek medical assistance, subjective improvements would encourage people to comply with therapeutic interventions. Additional controlled studies including subjective and objective measures of sleep in these patient groups are required.

Examining objective data, fit individuals seem to have longer sleep duration, shorter sleep onset latencies [60,61] and higher levels of SWS [61,62] than unfit subjects. In the reverse situation, the sleep of highly proficient athletes was assessed when they were not training due to injury or other reasons and later when fit. When fit, these athletes tended to sleep longer and had elevated SWS levels independent of their fitness status when compared with non-athletic, sedentary males [45]. Thus more athletic individuals, whether in training or not, may be better sleepers than their non-athletic counterparts. Indeed, it has been suggested that sleep may not vary as a function of aerobic fitness, but is indicative of some enduring characteristic in the subjects tested; sleep is therefore unlikely to be altered by improving cardiorespiratory fitness. To investigate this, the same individuals should be studied at different levels of fitness as has been done in the following studies.

Of the seven longitudinal studies published to date [3,32,58,59,63–65], three were conducted on “good sleepers” and included polysomnographic recordings [63–65]. Participation in a physical training program with improved fitness resulted in improved

sleep quality in eight male army recruits on an 18-week training programme [63]. The dramatic improvements in sleep, and particularly increased SWS, were noted when comparing the start to the middle and end of 18-weeks of training [63]. Considering the military nature of the recruits' environment, with factors such as profound stress and sleep deprivation, this finding has to be treated cautiously. A study of 30 older men and women (mean age 66 years) on a 6-month aerobic training programme showed enhanced SWS but no effect on overall subjective and objective sleep quality compared to pre-training [65]. A second group of 21 healthy seniors on a 6-month stretching/flexibility programme showed no change in VO_2max or sleep measures compared to pre-training; no condition by time comparison between the two training modalities was done [65]. In contrast, no effect of fitness on sleep was found in nine young women who participated in a 12-week endurance-training programme [64]. There is still no clear evidence whether improved fitness *per se* does indeed facilitate sleep in good sleepers. More promising results have been reported in randomized, controlled prospective studies of individuals with disturbed sleep [3,32,58,59] as outlined above.

Based on data collated from 12 studies, on the effects of chronic exercise or improved fitness on sleep [31], positive effect sizes for TST (ES of 0.94 from six studies) and SWS (ES of 0.43 from nine studies) indicated more sleep of deeper quality. Negative effect sizes showed a shorter SOL (ES of -0.45 from seven studies), less REM sleep (ES of -0.57 from five studies) and time awake (ES of -0.4 from six studies) [31]. It should be noted however, that for this meta-analysis [31] cross-sectional and prospective studies were combined. Furthermore, some recording nights were preceded by exercise during the day whereas others were not, confounding interpretation.

Mode of exercise

Some evidence suggests that chronic endurance exercise might be more likely to enhance SWS compared with other modes of exercise. Trinder *et al.* [52] described the sleep of long distance runners, athletes performing mixed aerobic and anaerobic power training, power weightlifters and sedentary individuals. Endurance athletes had the highest levels of SWS while the power-trained group had the lowest.

Light exposure

There is compelling evidence that chronic exposure to bright light can enhance sleep [66]. The results of Guilleminault *et al.* [59] suggest that the effect of exposure to light may be more powerful than those associated with exercise. In addition to the training and sleep-hygiene education treatments discussed earlier [59], individuals with psychophysiological insomnia were randomly assigned to a third treatment which included sleep hygiene education and light therapy. The light therapy involved patients sitting in front of a bright light box (3000 lux) for 45 min a day, beginning 5 min after awakening. Wrist activity measurements taken the week before and the week after each 4-week protocol indicated that the exercise protocol elicited an improvement in sleep time of 17 min per night, whereas sleep hygiene alone resulted in a decrease in sleep time of 3 min per night. The most impressive effect was that the bright light condition increased TST by 54 min. In a randomized controlled trial of 120 patients with atypical depression

(prolonged sleep, weight gain, carbohydrate craving) conducted in Finland during winter, supervised fitness training combined with exposure to bright light (2500–4000 lux) resulted in greater relief from their symptoms of depression and more vitality than when the training was conducted in ordinary room light (400–600 lux) and compared to relaxation training [33]. Thus exposure to bright light is likely to contribute to any sleep [8] and mood-promoting [33] effects of exercise performed outdoors.

Age and gender

As stated earlier, the meta-analysis by Kubitz *et al.* [31] reported significant moderating effects of age and gender on the effects of chronic exercise on sleep. Older, fit men (60–72 years) have been found to have shorter sleep onset latencies, less WASO, more total slow-waves and higher sleep efficiency than sedentary controls [61]. Once again, these data are limited by a low number of studies and small sample sizes.

Overtraining

The overtraining syndrome may occur following chronic, intense levels of training, and is characterized by such physiological and psychological markers of distress as compromised immune system, muscular fatigue, hypercortisolaemia and depression that may rise to clinical levels [51]. There is anecdotal evidence that overtraining disrupts sleep [67] but empirical evidence has been limited [51]. Although it was apparent that the swimmers in this latter study did not experience the full syndrome of overtraining described by others, when their swimming training was reduced to low volume anaerobic exercise, SWS time was also reduced and coincided with the poorest mood scores. This latter finding, of altered mood with varying training intensity, highlights another factor that has largely been ignored in exercise and sleep studies but which deserves closer attention in future studies.

Practice Points

1. Chronic exercise appears to increase TST and SWS (i.e. more sleep of deeper quality) and decrease sleep onset latency, REM sleep and WASO.
2. Beneficial effects of chronic exercise are more apparent in older populations and people with sleep complaints.
3. Improvements in self-rated sleep quality have been found following 16-weeks of moderate-intensity endurance training in older sedentary men and women who reported moderate sleep complaints [58]. In middle-aged to elderly subjects, a reduced likelihood of having a disorder in maintaining sleep and of having a sleep complaint has been associated with regular weekly activity.
4. Overtraining may lead to increased fatigue and more disturbed sleep.
5. The beneficial effects of exercise on sleep, even if only moderate, should not be underestimated particularly in patients with insomnia. Patients often need a sense of control over their sleep and engaging in regular aerobic activity may help them to break the cycle of poor sleep hygiene and insomnia.

Potential mechanisms underlying interaction between exercise and sleep

Circadian rhythms and bright light

Appropriately timed exposure to circadian phase-shifting stimuli both normalizes circadian phase and enhances sleep [66,68]. The discrepancy between anecdotal and experimental evidence that exercise promotes sleep combined with the different findings between exercise outdoors or in a laboratory or gym facility might partly be explained by an illumination mechanism [8]. Whereas the average adult receives only about 20 min of daily exposure to light exceeding 2500 lux, it is reasonable to suppose that many people who exercise outdoors (e.g. 1 h per day at 10 000 lux) receive at least 10 times this much [8]. Bright light might promote sleep by at least two mechanisms, specifically via antidepressant effects and via circadian phase-shifting effects. Therefore, it is plausible that exercise in well-lit conditions may improve sleep for shift workers, air travelers, patients with atypical depression, and older individuals with abnormal circadian timing.

Research Agenda

Possible interactions between light and exercise in altering sleep and circadian rhythms need to be explored. There is a need to determine whether there is a phase response curve for exercise and sleep akin to that known for light.

Strenuous exercise and the cytokine response

As described earlier, although it has been presumed that very long, strenuous exercise might promote sleep, it is equally plausible that such exercise might disrupt sleep due to muscle damage and pain. There is evidence that skeletal muscle and tissue damage stimulates production of inflammatory cytokines, including interleukin (IL)-6, IL-1 and tumour necrosis factor (TNF). Musculoskeletal damage is induced mostly after eccentric exercise, such as braking to oppose the rotation of bicycle pedals or downhill running, rather than concentric exercise (such as normal cycling) [69]. Increased levels of cytokines may, in turn, contribute to the delayed onset of muscle soreness 24 h after eccentric exercise [69]. The magnitude of the increase in cytokines is also probably related to the duration and intensity of exercise [69] as well as the level of fitness. The involvement of some of the cytokines in sleep regulation [70] suggests a role for these humoral factors in post-exercise sleep, and requires further investigation in conjunction with exercise.

Conclusions

Self-report studies have indicated that moderate and regular physical activity has therapeutic and sleep promoting benefits [3,4]. Numerous variables in available research data confound a precise interpretation of the effect of exercise on sleep [5–7,31,36]. Exercise does not always dramatically and directly affect sleep, yet the occasional

positive outcomes indicate that, under certain conditions, exercise may have beneficial effects. The most beneficial effect seems to come from improved fitness with aerobic endurance training and acute exercise that lasts for more than an hour. Conversely, exhaustive exercise of high-intensity and long duration is disruptive to sleep, decreasing REM sleep and increasing wakefulness.

Most studies relating to the effect of exercise on sleep have been performed in normal healthy young adults who are "good sleepers", which may limit the observed effects on sleep. In clinical populations, self-report data indicates that regular exercise is a useful modality in treating disorders of initiating and maintaining sleep, as well as complaints of poor sleep quality. Crucially, exercise needs to be prescribed on an individual basis and approached in a safe way to make it rewarding to the participant and to promote physical well-being [9]. Even small improvements in sleep with exercise should not be disregarded, particularly in patients with insomnia since engaging in regular aerobic activity may help them to break the cycle of insomnia. While it is plausible that exercise has a unique behavioural effect on sleep, the effects on sleep in untrained and clinical populations have yet to be clearly established.

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References

- 1 Hauri PJ. Consulting about insomnia: A method and some preliminary data. *Sleep* 1993; **16**: 344–350.
- 2 Lavie P. *The enchanted world of sleep*. New Haven, CT: Yale University Press, 1996.
- *3 Sherrill DL, Kotchou K, Quan SF. Association of physical activity and human sleep disorders. *Arch Int Med* 1998; **158**: 1894–1898.
- 4 Vuori I, Urponen H, Hasan J, Partinen M. Epidemiology of exercise effects on sleep. *Acta Physiol Scand* 1988; **133** (Suppl. 574): 3–7.
- *5 Trinder J, Montgomery I, Paxton SJ. The effect of exercise on sleep: the negative view. *Acta Physiol Scand* 1988; **133** (Suppl. 574): 14–21.
- *6 Driver HS, Taylor SR. Sleep disturbances and exercise. *Sports Med* 1996; **21**: 1–6.
- *7 Youngstedt SD, O'Connor PJ, Dishman RK. The effects of acute exercise on sleep: a quantitative synthesis. *Sleep* 1997; **20**: 203–214.
- 8 Youngstedt SD. Does exercise truly enhance sleep? *The Physician and Sports Med* 1997; **25**: 72–82.
- 9 Appenzeller O. Neurology of endurance training. In: Appenzeller O (ed.). *Sports Medicine. Fitness. Training. Injuries*. Baltimore, USA: Urban & Schwarzenberg, 1988, pp. 35–71.
- 10 Morin CM, Hauri PJ, Espie CA *et al.* Nonpharmacologic treatment of chronic insomnia. *Sleep* 1999; **22**: 1134–1156.
- 11 McGinty D, Szymusiak R. Keeping cool, a hypothesis about the mechanisms and functions of slow-wave sleep. *Trends Neurosci* 1990; **13**: 480–487.
- 12 Adam K, Oswald I. Protein synthesis, bodily renewal and the sleep-wake cycle. *Clin Sci* 1983; **65**: 561–567.

* The most important references are denoted by an asterisk.

- 13 Berger RJ, Phillips NH. Comparative aspects of energy metabolism, body temperature and sleep. *Acta Physiologica Scandinavica* 1988; **133** (Suppl 574): 21–28.
- 14 Murphy PJ, Campbell SS. Nighttime drop in body temperature: a physiological trigger for sleep onset? *Sleep* 1997; **20**: 505–511.
- 15 Krauchi K, Cajochen C, Werth E, Wirz-Justice A. Warm feet promote the rapid onset of sleep. *Nature* 1999; **401**: 36–37.
- 16 Glotzbach SF, Heller HC. Temperature Regulation. In: Kryger MH, Roth T, Dement WC (eds). *Principles and Practice of Sleep Medicine*. Philadelphia: W.B. Saunders Co. 1994, pp. 260–275.
- 17 Horne JA, Moore VJ. Sleep EEG effects of exercise with and without additional body cooling. *Electroenceph Clin Neurophysiol* 1985; **60**: 33–38.
- 18 Shapiro CM, Allan M, Driver HS, Mitchell D. Thermal load alters sleep. *Biol Psychiatry* 1989; **26**: 736–740.
- 19 Deboer T. Brain temperature dependent changes in the electroencephalogram power spectrum of humans and animals. *J Sleep Res* 1998; **7**: 254–262.
- 20 Saltin B, Hermansen L. Esophageal, rectal, and muscle temperature during exercise. *J Appl Physiol* 1966; **21**: 1757–1762.
- 21 O'Connor PJ, Breus MJ, Youngstedt SD. Exercise-induced increase in core temperature does not disrupt a behavioral measure of sleep. *Physiol Behav* 1998; **64**: 213–217.
- 22 Youngstedt SD, Kripke DF, Elliott JA. Is sleep disturbed by vigorous late-night exercise? *Med Sci Sports Exerc* 1999; **31**: 864–869.
- 23 Driver HS, Shulman I, Baker FC, Buffenstein R. An altered energy content of the evening meal alters nocturnal body temperature but not sleep. *Physiol Behav* 1999; **000068**: 17–23.
- 24 Jennings RJ, Reynolds CFI, Houck PR *et al.* Age and sleep modify finger temperature responses to facial cooling. *Gerontology* 1993; **48**: M108–M116.
- 25 Gilbert SS, Cameron J, van den Heuvel C, Kennaway DJ, Dawson D. Peripheral heat loss: A predictor of the hypothermic response to daytime melatonin in young and older adults. *Physiol Behav* 1999; **66**: 365–370.
- 26 Zepelin H. Mammalian sleep. In: Kryger MH, Roth T, Dement WC (eds). *Principles and Practice of Sleep Medicine*. Philadelphia: W.B. Saunders Co., 1994, pp. 69–80.
- 27 Rechtschaffen A. Current perspectives on the function of sleep. *Perspectives in Biology and Medicine* 1998; **41**: 359–390.
- 28 Ryback RS, Lewis OF. Effects of prolonged bedrest on EEG sleep patterns in young, healthy volunteers. *Electroencephalogr Clin Neurophysiol* 1971; **31**: 395–399.
- 29 Karklin A, Driver HS, Buffenstein R. Restricted energy intake affects nocturnal body temperature and sleep patterns. *Am J Clin Nutr* 1994; **59**: 346–349.
- 30 Shapiro CM, Driver HS. Stress and sleep. In: Roussel B, Jouvet M, (eds). Proceedings of the 27th DRG Seminar: *Sleep and its implications for the military*. Lyon, France: A.C.M.E.L., 1988; 133–146.
- *31 Kubitz KA, Landers DM, Petruzello SJ, Han M. The effects of acute and chronic exercise on sleep: a meta-analytic review. *Sports Med* 1996; **21**: 277–291.
- 32 Singh NA, Clements KM, Fiatarone MA. A randomized controlled trial of the effect of exercise on sleep. *Sleep* 1997; **20**: 95–101.
- 33 Partonen T, Leppamaki S, Hurme J, Lonnqvist J. Randomized trial of physical exercise alone or combined with bright light on mood and health-related quality of life. *Psychological Med* 1998; **28**: 1359–1364.
- 34 Netzer N, Lormes W, Giebelhaus V *et al.* Physical training of patients with sleep apnea. *Pneumologie* 1997; **51** (Suppl. 3): 779–782.
- 35 Driver HS, Meintjes AF, Rogers GG, Shapiro CM. Submaximal exercise effects on sleep patterns in young women before and after an aerobic training programme. *Acta Physiol Scand* 1988; **133** (Suppl. 574): 8–14.
- 36 Taylor SR, Driver HS. Is sleep affected by physical exercise and fitness? *Crit Rev Physical Rehab Med* 1995; **7**: 131–145.
- 37 Suskin N, Ryan G, Fardy J, Clarke H, McKelvie R. Clinical workload decreases the level of aerobic fitness in housestaff physicians. *J Cardiopulmon Rehabil* 1998; **18**: 216–220.
- 38 Lichstein KL, Means MK, Noe SL, Aguillard RN. Fatigue and sleep disorders. *Behav Res Ther* 1997; **35**: 733–740.
- 39 Leproult R, van Reeth O, Byrne MM, Sturis J, Van Cauter E. Sleepiness, performance, and neuroendocrine function during sleep deprivation: Effects of exposure to bright light or exercise. *J Biol Rhythms* 1997; **12**: 245–258.
- 40 Horne JA, Foster SC. Can exercise overcome sleepiness? *Sleep Res* 1995; **24A**: 437.

- *41 Horne JA. The effects of exercise on sleep: a critical review. *Biol Psychol* 1981; **12**: 241–290.
- 42 Horne JA, Staff LHE. Exercise and sleep: body-heating effects. *Sleep* 1983; **6**: 36–46.
- 43 Bunnell DE, Bevier W, Horvath SM. Effects of exhaustive exercise on the sleep of men and women. *Psychophysiology* 1983; **20**: 50–58.
- 44 Paxton SJ, Montgomery I, Trinder J *et al*. Sleep after exercise of variable intensity in fit and unfit subjects. *Aust J Psychol* 1982; **34**: 289–296.
- 45 Paxton SJ, Trinder J, Montgomery I. Does aerobic fitness affect sleep? *Psychophysiol* 1983; **20**: 320–324.
- 46 Shapiro CM, Bortz R, Mitchell D *et al*. Slow wave sleep: A recovery period after exercise. *Science* 1981; **214**: 1253–1254.
- 47 Torsvall L, Akerstedt T, Lindbeck G. Effects on sleep stages and EEG power density of different degrees of exercise in fit subjects. *Electroencephalogr Clin Neurophysiol* 1984; **57**: 347–353.
- 48 Montgomery I, Trinder J, Paxton S *et al*. Sleep disruption following a marathon. *J Sports Med* 1985; **25**: 69–73.
- 49 Driver HS, Rogers GG, Mitchell D, Borrow SJ, Allen M, Luus HG, Shapiro CM. Prolonged endurance exercise and sleep disruption. *Med Sci Sports Exerc* 1994; **26**: 903–907.
- 50 Bencá RM, Okawa M, Uchiyama M *et al*. Sleep and mood disorders. *Sleep Med Rev* 1997; **1**: 45–56.
- 51 Taylor SR, Rogers GG, Driver HS. Effects of training volume on sleep, psychological, and selected physiological profiles of elite female swimmers. *Med Sci Sports Exerc* 1997; **29**: 688–693.
- 52 Trinder J, Paxton SJ, Montgomery I, Fraser G. Endurance as opposed to power training: their effect on sleep. *Psychophysiol* 1985; **22**: 668–673.
- 53 Montgomery I, Trinder J, Paxton S *et al*. Physical exercise and sleep: the effect of the age and sex of the subjects and type of exercise. *Acta Physiol Scand* 1988; **133** (Suppl. 574): 36–40.
- 54 Van Reeth O, Sturis J, Byrne MM, Blackman JD, L'Hermite-Baleriaux M, Leproult R, Oliner R, Refetoff S, Turek FW, Van Crauter E. Nocturnal exercise phase delays circadian rhythms of melatonin and thyrotropin secretion in normal men. *Am J Physiol* 1994; **266**: E964–E974.
- 55 Driver HS, Baker FC. Menstrual factors and sleep. *Sleep Med Rev* 1999; **3**: 219–228.
- 56 Ancoli-Israel S, Poceta JS, Stepnowsky C, Martin J, Gehrman P. Identification and treatment of sleep problems in the elderly. *Sleep Med Rev* 1997; **1**: 13–17.
- 57 Montgomery I, Trinder J, Paxton SJ. Aerobic fitness and exercise: effects on the sleep of younger and older adults. *Aust J Psychol* 1987; **39**: 259–272.
- *58 King AC, Oman RF, Brassington GS, Bliwise DL, Haskell WL. Moderate-intensity exercise and self-rated quality of sleep in older adults. *JAMA* 1997; **227**: 32–37.
- 59 Guilleminault C, Clerk A, Black J, Labanowski M, Pelayo R, Claman D. Nondrug treatment trials in psychologic insomnia. *Ann Intern Med* 1995; **155**: 838–844.
- 60 Montgomery I, Trinder J, Paxton SJ. Energy expenditure and total sleep time: effect of physical exercise. *Sleep* 1982; **5**: 159–169.
- *61 Edinger JD, Morey MC, Sullivan RJ, Higginbotham MB, Marsh GR, Dailey DS, McCall WV. Aerobic fitness, acute exercise and sleep in older men. *Sleep* 1993; **16**: 351–357.
- 62 Baekeland F, Lasky R. Exercise and sleep patterns in college athletes. *Perceptual and Motor Skills* 1966; **23**: 1203–1207.
- 63 Shapiro CM, Warren PM, Trinder J *et al*. Fitness facilitates sleep. *Eur J Appl Physiol* 1984; **53**: 347–353.
- 64 Meintjes AF, Driver HS, Shapiro CM. Improved physical fitness failed to alter the EEG patterns of sleep in young women. *Eur J Appl Physiol* 1989; **59**: 123–127.
- 65 Vitiello MV, Prinz PN, Schwartz RS. The slow wave sleep of healthy older men and women is enhanced with improved aerobic fitness. *J Sleep Res* 1994; **3** (Suppl. 1): 270.
- 66 Campbell SS, Dawson D, Anderson MW. Alleviation of sleep maintenance insomnia with timed exposure to bright light. *J Am Geriatr Soc* 1993; **41**: 829–836.
- 67 Budgett R. Overtraining syndrome. *Br J Sports Med* 1990; **24**: 231–236.
- 68 Dawson D, Armstrong SM. Chronobiotic-drugs that shift rhythms. *Pharmacol Ther* 1996; **69**: 15–36.
- 69 Pedersen BK, Ostrowski K, Rohde T, Bruunsgaard H. The cytokine response to strenuous exercise. *Can J Physiol Pharmacol* 1998; **76**: 505–511.
- 70 Dickstein JB, Moldofsky H. Sleep, cytokines and immune function. *Sleep Med Rev* 1998; **2**: 213–229.